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NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	FEB 25	CA/CAPLUS - Russian Agency for Patents and Trademarks (ROSPATENT) added to list of core patent offices covered
NEWS	4	FEB 28	PATDPAFULL - New display fields provide for legal status data from INPADOC
NEWS	5	FEB 28	BABS - Current-awareness alerts (SDIs) available
NEWS	6	FEB 28	MEDLINE/LMEDLINE reloaded
NEWS	7	MAR 02	GBFULL: New full-text patent database on STN
NEWS	8	MAR 03	REGISTRY/ZREGISTRY - Sequence annotations enhanced
NEWS	9	MAR 03	MEDLINE file segment of TOXCENTER reloaded
NEWS	10	MAR 22	KOREAPAT now updated monthly; patent information enhanced
NEWS	11	MAR 22	Original IDE display format returns to REGISTRY/ZREGISTRY
NEWS	12	MAR 22	PATDPASPC - New patent database available
NEWS	13	MAR 22	REGISTRY/ZREGISTRY enhanced with experimental property tags
NEWS	14	APR 04	EPFULL enhanced with additional patent information and new fields
NEWS	15	APR 04	EMBASE - Database reloaded and enhanced
NEWS	16	APR 18	New CAS Information Use Policies available online
NEWS	17	APR 25	Patent searching, including current-awareness alerts (SDIs), based on application date in CA/CAplus and USPATFULL/USPAT2 may be affected by a change in filing date for U.S. applications.
NEWS	18	APR 28	Improved searching of U.S. Patent Classifications for U.S. patent records in CA/CAplus
NEWS	19	MAY 23	GBFULL enhanced with patent drawing images
NEWS	20	MAY 23	REGISTRY has been enhanced with source information from CHEMCATS
NEWS	21	MAY 26	STN User Update to be held June 6 and June 7 at the SLA 2005 Annual Conference

NEWS EXPRESS JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

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FILE 'HOME' ENTERED AT 15:44:21 ON 31 MAY 2005

=> file hcaplus
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'HCAPLUS' ENTERED AT 15:44:28 ON 31 MAY 2005
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FILE COVERS 1907 - 31 May 2005 VOL 142 ISS 23
FILE LAST UPDATED: 30 May 2005 (20050530/ED)

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This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> s prostacyclin () mimetic?
         10339 PROSTACYCLIN
           277 PROSTACYCLINS
         10414 PROSTACYCLIN
                 (PROSTACYCLIN OR PROSTACYCLINS)
          8197 MIMETIC?
L1          83 PROSTACYCLIN (W) MIMETIC?

=> s l1 and chronic? () inflammation
        194520 CHRONIC?
        132568 INFLAMMATION
          1832 INFLAMMATIONS
        133296 INFLAMMATION
                 (INFLAMMATION OR INFLAMMATIONS)
          3647 CHRONIC? (W) INFLAMMATION
L2           0 L1 AND CHRONIC? (W) INFLAMMATION

=> s l1 and inflammation
        132568 INFLAMMATION
          1832 INFLAMMATIONS
        133296 INFLAMMATION
                 (INFLAMMATION OR INFLAMMATIONS)
L3           2 L1 AND INFLAMMATION

=> d l3, ibib abs, 1-2

L3   ANSWER 1 OF 2  HCAPLUS  COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER:        2003:164608  HCAPLUS
```

DOCUMENT NUMBER: 139:17835
TITLE: Regulation of cyclooxygenase-2 expression by iloprost in human vascular smooth muscle cells Role of transcription factors CREB and ICER
AUTHOR(S): Debey, Svenja; Meyer-Kirchrath, Jutta; Schror, Karsten
CORPORATE SOURCE: Universitats Klinikum Dusseldorf, Institut fur Pharmakologie und Klinische Pharmakologie, Heinrich-Heine-Universitat, Dusseldorf, D-40225, Germany
SOURCE: Biochemical Pharmacology (2003), 65(6), 979-988
CODEN: BCPCA6; ISSN: 0006-2952
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Prostaglandin-endoperoxide synthase-2 (PGH-synthase) or cyclooxygenase-2 (COX-2) is inducible by a variety of stimuli, e.g. inflammatory mediators, growth factors and hormones and is believed to be responsible for the majority of inflammatory prostanoid production Moreover, it has been demonstrated that COX-2 contributes substantially to prostacyclin-synthesis in patients with atherosclerosis. In this study, we demonstrate an up-regulation of COX-2 mRNA, protein and product formation by the **prostacyclin-mimetic** iloprost in human vascular smooth muscle cells (hSMC). COX-2 mRNA expression was induced transiently between 1 and 6 h and returned to basal levels after 16 h of iloprost stimulation. COX-2 protein was induced concomitantly between 3 and 6 h of iloprost stimulation. This was accompanied by an increase in PGI2 formation. Forskolin, a direct activator of adenylyl cyclase, and dibutyryl cAMP, a cell-permeable cAMP analog-induced COX-2 mRNA, suggesting a cAMP-dependent COX-2 expression in hSMC. Iloprost-induced COX-2 protein expression and PGI2 formation was synergistically elevated by co-stimulation with the phorbol-ester PMA (phorbol-12-myristate-13-acetate). It is concluded, that the observed up-regulation of COX-2 with subsequent release of newly synthesized PGI2 and the synergistic effect of iloprost and phorbol-ester on PGI2 formation provide a pos. feedback of prostaglandins on their own synthesizing enzyme. This might be important for control of hSMC proliferation, migration and differentiation as well as inhibition of platelet aggregation.

REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2000:776145 HCAPLUS
DOCUMENT NUMBER: 134:982
TITLE: The **prostacyclin-mimetic** cicaprost inhibits endogenous endothelin-1 release from human pulmonary artery smooth muscle cells
AUTHOR(S): Wort, S. John; Mitchell, Jane A.; Woods, Mandy; Evans, Timothy W.; Warner, Timothy D.
CORPORATE SOURCE: Unit of Critical Care, Imperial College School of Medicine, Royal Brompton Hospital, London, SW3 6NP, UK
SOURCE: Journal of Cardiovascular Pharmacology (2000), 36(5, Suppl. 1), S410-S413
CODEN: JCPCDT; ISSN: 0160-2446
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB There is increasing evidence supporting a role for endothelin-1 (ET-1) in human pulmonary hypertension. The aim of this study was to determine the relative roles of human pulmonary microvascular endothelial cells (HPMVE) and human pulmonary artery smooth muscle (HPASM) cells to produce ET-1 under inflammatory conditions and to investigate further possible control mechanisms of ET-1 production by HPASM. Although HPMVE cells produced more ET-1 than HPASM when cultured with fetal calf serum (FCS) alone and after

treatment with cytokines; HPASM produced significant amts. of ET-1 after stimulation with cytokines. Cytokine-stimulated increase in ET-1 production by HPASM was inhibited by cicaprost, a prostacyclin analog, and other agents that are known to increase intracellular cAMP. Cicaprost also inhibited proliferation of HPASM in response to FCS lending support to the theory that part of the clin. benefit seen in long-term treatment with prostacyclin in pulmonary hypertension may be a result of inhibition of ET-1 production in these cells.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

```
=> file prostacyclin (PG12)
'PROSTACYCLIN' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available.  If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.
ENTER A FILE NAME OR (IGNORE):end

=> file ph12
'PH12' IS NOT A VALID FILE NAME
SESSION CONTINUES IN FILE 'HCAPLUS'
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available.  If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.

=> file pg12
'PG12' IS NOT A VALID FILE NAME
SESSION CONTINUES IN FILE 'HCAPLUS'
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available.  If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.

=> file prostacyclin 12
'PROSTACYCLIN' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available.  If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.
ENTER A FILE NAME OR (IGNORE):end

=> s PG 12 or prostacyclin 12
         50042 PG
          4179 PGS
         51954 PG
                 (PG OR PGS)
       1322220 12
            30 PG 12
                 (PG(W)12)
         10339 PROSTACYCLIN
           277 PROSTACYCLINS
         10414 PROSTACYCLIN
                 (PROSTACYCLIN OR PROSTACYCLINS)
       1322220 12
             1 PROSTACYCLIN 12
                 (PROSTACYCLIN(W)12)
L4           31 PG 12 OR PROSTACYCLIN 12

=> s l4 and review/dt
       1826960 REVIEW/DT
```

L5 1 L4 AND REVIEW/DT

=> d l5, ibib abs, 1

L5 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:47560 HCAPLUS
DOCUMENT NUMBER: 140:104312
TITLE: Pulmonary arterial hypertension: current therapies
AUTHOR(S): Delcroix, M.; Budts, W.; Corris, P. A.; Daenen, W.; Gewillig, M.; Naeije, R.; Olschewski, H.; Pepke-Zaba, J.; Seeger, W.; Vachiery, J-L.; Van Raemdonck, D.; Vizza, C. D.; Galie, N.; Kneussl, M.; Nicod, L.; Abramowics, M.; Adnot, S.; Barbera, J. A.; Boonstra A.; Dorfmueller, P.; Eddahibi, S.; Ghofrani, H.; Herve, P.; Gruenig, E.; Hoeper, M. M.; Janssens, S.; MacLean, M. R.; Morrell, N. W.; Peacock, A.; Sitbon, O.; Trembath, R. C.; Verleden, G. M.
CORPORATE SOURCE: Pulmonary Arterial Hypertension, University Hospital, Louvain, Belg.
SOURCE: European Respiratory Monograph (2003), Volume Date 2004, 9(27), 57-83
CODEN: EURMF6; ISSN: 1025-448X
PUBLISHER: European Respiratory Society Journals Ltd.
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review. Pulmonary arterial hypertension (PAH) is a severe disease with poor prognosis and a poor quality of life due to reduced exercise capacity. Considerable improvement has been obtained during the last 10 yrs with the progressive introduction of pulmonary vasodilator prostaglandin (**PG**)**12** analogs and anti-endothelins. This chapter describes these recent therapeutic advances. It will also include tech. and surgical therapeutic options developed to alleviate, to cure or to prevent pulmonary hypertension.
REFERENCE COUNT: 114 THERE ARE 114 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s prostacyclin? () inflammation?
10452 PROSTACYCLIN?
133297 INFLAMMATION?
L6 8 PROSTACYCLIN? (W) INFLAMMATION?

=> s l6 and review/dt
1826960 REVIEW/DT
L7 1 L6 AND REVIEW/DT

=> d l7, ibib abs, 1

L7 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2002:850622 HCAPLUS
DOCUMENT NUMBER: 138:134496
TITLE: Update: high altitude pulmonary edema
AUTHOR(S): Baertsch, Peter; Swenson, Erik R.; Maggiorini, Marco
CORPORATE SOURCE: Department of Internal Medicine, Division of Sports Medicine, University of Heidelberg, Heidelberg, Germany
SOURCE: Advances in Experimental Medicine and Biology (2001), 502(Hypoxia), 89-106
CODEN: AEMBAP; ISSN: 0065-2598
PUBLISHER: Kluwer Academic/Plenum Publishers
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review. Recent high altitude studies with pulmonary artery (PA) catheterization and broncho-alveolar lavage (BAL) in early high altitude pulmonary edema(HAPE) have increased our understanding of the pathogenetic sequence in HAPE. High preceding PA and pulmonary capillary pressures lead to a non-inflammatory leak of the alveolar-capillary barrier with egress of red cells, plasma proteins and fluid into the alveolar space. The mechanisms accounting for an increased capillary pressure remain speculative. The concept that hypoxic pulmonary vasoconstriction (HPV) is uneven so that regions with less vasoconstriction are over-perfused and become edematous remains compelling but unproved. Also uncertain is the role and extent of pulmonary venoconstriction. With disruption of the normal alveolar-capillary barrier, some individuals may later develop a secondary inflammatory reaction. A high incidence of preceding or concurrent respiratory infection in children with HAPE has been used to support a causative role of inflammation in HAPE. However, alternatively even mild HPV may simply lower the threshold at which inflammation-mediated increases in alveolar capillary permeability cause significant fluid flux into the lung. Other major questions to be addressed in future research are: (1) What is the mechanism of exaggerated hypoxic pulmonary vasoconstriction. Is there a link to primary pulmonary hypertension. Several observations suggest that susceptibility to HAPE is associated with endothelial dysfunction in pulmonary vessels. This has not yet been studied adequately. (2) What is the nature of the leak. Is there structural damage, i.e. stress failure, or does stretch cause opening of pores. (3) What is the pathophysiol. significance of a decreased sodium and water clearance across alveolar epithelial cells in hypoxia. (4) What is the role of exercise. Do HAPE-susceptible individuals develop pulmonary edema when exposed to hypoxia without exercise. Answers to these questions will increase our understanding of the pathophysiol. of HAPE and also better focus research on the genetic basis of susceptibility to HAPE.

REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 15:44:21 ON 31 MAY 2005)

FILE 'HCAPLUS' ENTERED AT 15:44:28 ON 31 MAY 2005

L1 83 S PROSTACYCLIN () MIMETIC?
L2 0 S L1 AND CHRONIC? () INFLAMMATION
L3 2 S L1 AND INFLAMMATION
L4 31 S PG 12 OR PROSTACYCLIN 12
L5 1 S L4 AND REVIEW/DT
L6 8 S PROSTACYCLIN? () INFLAMMATION?
L7 1 S L6 AND REVIEW/DT

=> s l1 and review/dt
1826960 REVIEW/DT
L8 8 L1 AND REVIEW/DT

=> d l8, ibib abs, 1-8

L8 ANSWER 1 OF 8 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1998:179513 HCAPLUS
DOCUMENT NUMBER: 128:289544
TITLE: Gastroprotective agents for the prevention of NSAID-induced gastropathy
AUTHOR(S): Ares, Jeffrey J.; Outt, Pamela E.
CORPORATE SOURCE: Health Care Res. Center, Procter & Gamble Pharmaceuticals, Mason, OH, 45040, USA
SOURCE: Current Pharmaceutical Design (1998), 4(1), 17-36

CODEN: CPDEFP; ISSN: 1381-6128
PUBLISHER: Bentham Science Publishers
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English

AB A review with 173 refs. Over 30 million people in the world take non-steroidal anti-inflammatory drugs (NSAIDs). A large percentage of these individuals will develop gastric ulcers and related complications, a condition known as "NSAID gastropathy". NSAID gastropathy differs from classic peptic ulcer disease in many ways, and traditional peptic ulcer therapy is largely ineffective in preventing NSAID-induced gastropathy. The prostaglandin misoprostol has been shown to be effective and is approved for the prevention of NSAID gastropathy. However, misoprostol has side effects that limit its general use. For this reason, considerable effort throughout the 1990's has focused on the identification of new gastroprotective mols. Some synthetic studies have been aimed at the preparation of new prostaglandins, **prostacyclin mimetics**, and thromboxane antagonists. New histamine H2 receptor antagonists have also been developed which, unlike cimetidine or ranitidine, now appear to couple true gastroprotective activity with antisecretory properties. One new H2 antagonist, ebrotidine, has shown clin. utility in preventing NSAID gastropathy. Many other types of structures (flavonoids, peptides, terpenoids, xanthines, others), as well as compds. displaying certain pharmacol. actions (5-hydroxytryptamine receptor binding, adrenergic receptor binding, mast cell stabilization, others) have been linked in some way to gastroprotection. This article reviews many of these recent gastroprotection findings, with emphasis on these of potential use for prevention of NSAID gastropathy.

REFERENCE COUNT: 173 THERE ARE 173 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 2 OF 8 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1998:69912 HCAPLUS
DOCUMENT NUMBER: 128:188184
TITLE: Acetylsalicylic acid in comparison with other thrombocyte function inhibitors - mechanisms of action
AUTHOR(S): Tschoepe, D.; Schwippert, B.
CORPORATE SOURCE: Arbeitsgruppe "Zellulare Hamostase und Klinische Angiologie", Diabetes-Forschungsinstitut an der Heinrich-Heine-Univ., Dusseldorf, D-40225, Germany
SOURCE: Acetylsalicylsaeure im Kardiovaskulaeren System (1996), 62-88. Editor(s): Schroer, K.; Breddin, H. K. Birkhaeuser: Basel, Switz.
CODEN: 65OCAH
DOCUMENT TYPE: Conference; **General Review**
LANGUAGE: German

AB A review with 82 refs. Acetylsalicylic acid is the "gold standard" for reducing the incidence and improving the outcome of acute thrombotic events in arteries. Acetylsalicylic acid belongs to the small group of drugs with established beneficial effects on life expectancy in secondary prevention. Similar, though less well established, conclusions have also been drawn for primary prevention. This makes it difficult to design other, possibly more contemporary concepts of drug-induced inhibition of platelet function vs. the concept of inhibition of cyclooxygenase. Inhibition of platelet function is more than inhibition of thrombogenesis and also involves local vasomotor control, vascular remodeling and activation of addnl. blood cells, induced by platelet-derived mediators. The evaluation of the efficacy of inhibitors of platelet function, therefore, must consider these facts. This is also true for acetylsalicylic acid, where short-term effects need to be separated from long-term effects, and direct actions from indirect actions. The action mechanism involves the thromboxane signal and, therefore, is limited in short-term use. On the other hand, any change in cyclooxygenase

metabolites by acetylsalicylic acid may allow for addnl. effects, such as modulation of white cell function. More recently developed inhibitors of platelet function include ticlopidine, **prostacyclin mimetics** and antiadhesive compds., such as fibrinogen receptor antagonists. Fibrinogen receptor antagonists block platelet aggregation, i.e., the final common pathway of activated platelets. These highly potent compds. will not replace the more general concept of cyclooxygenase inhibition. However, data suggest that, like fibrinolytics, a combination of different antiplatelet strategies may result in an improved clin. efficacy.

REFERENCE COUNT: 82 THERE ARE 82 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 3 OF 8 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1998:20836 HCAPLUS

DOCUMENT NUMBER: 128:149603

TITLE: Prostanoid action on the human pulmonary vascular system

AUTHOR(S): Jones, Robert L.; Qian, Yue-Ming; Wong, Henry N. C.; Chan, Ho-Wai; Yim, Anthony P. C.

CORPORATE SOURCE: Department of Pharmacology, Chinese University of Hong Kong, Shatin, Peop. Rep. China

SOURCE: Clinical and Experimental Pharmacology and Physiology (1997), 24(12), 969-972
CODEN: CEXPB9; ISSN: 0305-1870

PUBLISHER: Blackwell Science Pty Ltd.

DOCUMENT TYPE: Journal; **General Review**

LANGUAGE: English

AB A review, with 27 refs. Four types of prostanoid receptor are present on pulmonary arterial vessels of man. Thromboxane (TP) receptors mediate constriction and are blocked by antagonists such as BAY u-3405, GR 32191 and EP 169. Prostaglandin (PG) EP3 receptors also mediate constriction, the agonist potency ranking being SC 46275>sulprostone>misoprostol≥ PGE2; this action needs to be borne in mind when PGE analogs are used therapeutically. Prostaglandin E2 causes relaxation in a few pulmonary artery prepns.: an EP2 receptor may be involved. Prostacyclin, acting through IP receptors, consistently produces relaxation and studies are in progress to determine the contribution made by K+-channel opening. Agonist potencies of stable prostacyclin analogs and non-prostanoid **prostacyclin mimetics**, such as BMY 45778 and the novel diphenylindole CU 23, on human pulmonary artery and platelets are well correlated. Interestingly, the non-prostanoid mimetics show persistent relaxant effects in vitro, which may be related to their high lipophilicities. Prostacyclin and iloprost are being used to treat severe pulmonary hypertension; further study of the pharmacodynamic and pharmacokinetic properties of other IP receptor agonists could produce improved therapy.

REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 4 OF 8 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1997:409643 HCAPLUS

DOCUMENT NUMBER: 127:76070

TITLE: Roles of vasodilatory prostaglandins in mitogenesis of vascular smooth muscle cells

AUTHOR(S): Schroer, Karsten; Weber, Artur-Aron

CORPORATE SOURCE: Institut fur Pharmakologie, Heinrich-Heine-Universitat Dusseldorf, Dusseldorf, D-40225, Germany

SOURCE: Agents and Actions Supplements (1997), 48(Prostaglandins and Control of Vascular Smooth Muscle Cell Proliferation), 63-91
CODEN: AASUDJ; ISSN: 0379-0363

PUBLISHER: Birkhaeuser

DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review, with 123 refs. Vasodilatory prostaglandins (PGI2, PGE1) and synthetic **prostacyclin mimetics** inhibit smooth muscle cell proliferation in vitro after stimulation by growth factors. Similar results are obtained in vivo after endothelial injury, suggesting that vasodilatory prostaglandins might also control smooth muscle cell proliferation in vivo. However, available data from clin. trials are conflicting and currently do not support the concept that these compds. might be successfully used to suppress excessive smooth muscle cell growth in response to tissue injury, specifically restenosis after PTCA. One possible explanation for these different results is an agonist-induced down-regulation of prostacyclin receptors in vascular smooth muscle cells. It is possible that enhanced endogenous prostacyclin biosynthesis, subsequent to induction of COX-2 and/or in relation to the formation of a neointima from media smooth muscle cells, might have a similar effect. There is still uncertainty regarding the cellular signal transduction pathways and their possibly complex interaction, although cAMP-dependent reactions are probably involved. In addition, vasodilatory prostaglandins might also interfere with the generation and action of other growth modulating factors, including PDGF, hepatocyte growth factor and nitric oxide. In conclusion, vasodilatory prostaglandins might be considered as growth modulating endogenous mediators in vascular smooth muscle cells.
REFERENCE COUNT: 123 THERE ARE 123 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1996:81463 HCAPLUS
DOCUMENT NUMBER: 124:106840
TITLE: Focus on prostacyclin and its novel mimetics
AUTHOR(S): Wise, Helen; Jones, Robert L.
CORPORATE SOURCE: Dep. Pharmacology, Chinese Univ., Shatin, Hong Kong
SOURCE: Trends in Pharmacological Sciences (1996), 17(1), 17-21
CODEN: TPHSDY; ISSN: 0165-6147
PUBLISHER: Elsevier Trends Journals
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review, with 52 refs. Prostacyclin (PGI2) has been traditionally regarded as an important regulator of hemostasis, mediating its effects through prostacyclin (IP) receptors coupled to adenylate cyclase. Recent evidence suggests, however, that IP receptor agonists can activate multiple signaling pathways via the same IP receptor. Moreover, IP receptor agonists have interesting actions outside of the cardiovascular system, even extending to the release of non-adrenergic non-cholinergic (NANC) transmitters from enteric neurons. Here, the authors highlight some of this new information on PGI2 and its receptors, describe the properties of some chemical novel PGI2 mimetics, and report on current therapeutic uses of PGI2.

L8 ANSWER 6 OF 8 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:793222 HCAPLUS
DOCUMENT NUMBER: 123:218463
TITLE: **Prostacyclin mimetics** with non-prostanoid structures
AUTHOR(S): Kondo, Kigen; Hamanaka, Nobuyuki
CORPORATE SOURCE: Minase Res. Inst., Ono Pharm. Co., Ltd., Osaka, 618, Japan
SOURCE: Nippon Yakurigaku Zasshi (1995), 106(3), 181-91
CODEN: NYKZAU; ISSN: 0015-5691
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: Japanese

AB A review, with 21 refs., on discovery of hydronaphthalene derivs. capable of interacting with prostanoid receptors, synthesis of prostacyclin agonists with non-prostanoid structures that show thromboxane synthase-inhibiting activity, and a trend in the development of prostacyclin agonists with non-prostanoid structure.

L8 ANSWER 7 OF 8 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1994:208659 HCAPLUS
DOCUMENT NUMBER: 120:208659
TITLE: Endothelial control of cell migration and proliferation
AUTHOR(S): Graf, H.
CORPORATE SOURCE: Schering Res. Lab., Berlin, 13342, Germany
SOURCE: European Heart Journal (1993), 14(Suppl. I), 183-6
CODEN: EHJODF; ISSN: 0195-668X
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB A review, with 31 refs., on transendothelial migration of monocytes and tumor cells and endothelial control of cell proliferation. The vascular endothelium was found to be involved in numerous physiol. and pathophysiol. processes and to act as a gating element in between cellular components of the blood and the adjacent tissue. In this respect, endothelial cells are especially important for the control of the extravasation of immune-competent cells and tumor cells and in tissue remodelling. These processes are guided by a network of intercellular recognition and transduction mechanisms to guarantee controlled, selective, target-directed action by extravasating cells and for complete functional remodelling of the adjacent tissue. Prostacyclin seems to play an important part in this endothelium-dependent intercellular crosstalk by modulating cell activation without altering the basal cellular mechanisms, as can be shown using stable **prostacyclin mimetics**, both in animal models and in cell culture.

L8 ANSWER 8 OF 8 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1993:74238 HCAPLUS
DOCUMENT NUMBER: 118:74238
TITLE: Interactions between arachidonic and eicosapentaenoic acids during their dioxygenase-dependent peroxidation
AUTHOR(S): Lagarde, M.; Vericel, E.; Croset, M.; Calzada, C.; Bordet, J. C.; Guichardant, M.
CORPORATE SOURCE: INSA, Lyon, Fr.
SOURCE: Prostaglandins, Leukotrienes and Essential Fatty Acids (1993), 48(1), 23-5
CODEN: PLEAEU; ISSN: 0952-3278
DOCUMENT TYPE: Journal; **General Review**
LANGUAGE: English
AB Eicosapentaenoic acid (EPA), a major polyunsatd. fatty acid of fish has been widely proposed as a potential nutrient for decreasing platelet-endothelial cell interactions and the subsequent atherogenesis and thrombogenesis. This is mainly based upon the decrease of arachidonic acid (AA) oxygenation into bioactive mols. like thromboxane A2. In addition, EPA may be oxygenated into its own active derivs. via cell dioxygenases. The authors discuss evidence for the requirement of specific peroxides, adequately provided by AA, to allow EPA to be oxygenated into its bioactive products like prostaglandin I3, a **prostacyclin mimetic**. The authors present some data that argue for a decreased basal AA dioxygenation (specific peroxidn.) by small concns. of EPA. The interactions between AA and EPA are then dual, EPA being able to counteract AA oxygenation whereas EPA requires AA to be efficiently oxygenated.

=>